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ERRATUM

M. Blau and Richard W. Compans, pages 281–289: It was reported that serial passage of measles virus in a polarized epithelial cell line caused a progressive change from a predominantly apical release pattern to a bidirectional pattern of release. To characterize further the alterations that were responsible for the observed change in the site of budding in the passaged measles virus, the HA gene was amplified by PCR from the 6th passage virus, and the sequence of the first 1427 nt which code for sequences in the external domain were determined. No differences between the deduced amino acid sequence and that reported previously for HA of the Edmondston strain (Alkhatib and Briedis, 1986, Virology 150:479–490) were observed. Because of the observed differences in several properties of the passaged virus, such as basolateral entry and a change in the CD46 binding requirement, this was a puzzling result. The passaged virus was previously examined by negative staining and thin section electron microscopy and it was found that only paramyxovirus particles could be detected. The possibility that another paramyxovirus might be present in the passaged stocks was therefore considered. Cells infected with the passaged virus stocks were examined by indirect immunofluorescence using antisera to parainfluenza viruses, which are being studied in the authors' laboratory. It was observed that human parainfluenza virus type 3 (PI3) virus antigen was present in increasing amounts upon passage, and cells infected with passages 3 or 4 of the "passaged measles virus" stocks showed intense fluorescence with PI3 antisera. The presence of the PI3 virus is presumably responsible for the higher titers observed upon passage (Fig. 2) as well as for the enhanced basolateral release. In an independent series of serial passages of PI3 virus in Vero C1008 cells, a change in the pattern of release from almost exclusively apical release in the original stock to approximately 25% basolateral release in the 4th passage was observed. No cross-reactivity was detected between the antisera used to detect measles virus HA protein and PI3 viral antigens. Thus, the immunofluorescence results previously shown in Fig. 4 represent the localization of the measles HA protein and not PI3 proteins. The observed changes in ability to down-regulate CD46 and in virus entry, from a preferential apical entry to a bidirectional entry pattern (Fig. 6), are also likely to be due to the presence of PI3 virus, rather than to a measles variant which does not require CD46. The sialic acid receptors for PI3 virus are known to be present on both surfaces of polarized epithelial cells (Fuller *et al.,* 1984, Cell 38:65–77). The authors regret that this error occurred and that it was not discovered until after publication.

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